4 Keay SD, Barlow R, Eley A, *et al.* The relation between immunoglobulin G antibodies to Chlamydia trachomatis and poor ovarian response to gonadotrophin stimulation before in vitro fertilisation. *Fertil Steril* 1998;**70**:214–8.
5 Gaudoin M, Rekha P, Morris A, *et al.* Bacterial vaginosis and post chlamydial infection are strongly and independently associated with tubal infertility but do not affect *in vitro* fertilization success rates. *Fertil Steril* 1999;**72**:730–2.
6 Conway D, Glazener CMA, Caul EO, *et al.* Chlamydial serology in fertile and infertile women. *Lancet* 1984;**1**:191–3.

Implications of random measurement error in studies adjusting for sexual behaviours

EDITOR,—In their recent review of methodological issues in sexual behaviour research, Fenton *et al*[1] provide a comprehensive overview of the major types of sexual behaviour research, the sources of measurement error which may affect such research, and different approaches to measuring various forms of measurement error. We would like to provide an important footnote on the implications of the poor measurement of sexual behaviours for drawing inferences from studies of sexually transmitted infections (STIs) which attempt to adjust for sexual behaviours in their analyses.

The role of systematic measurement errors in study design and analysis, as described by Fenton *et al*, is widely recognised. Given their impact on inferences of association, great care is taken in most studies to avoid these biases. The effects of random measurement error, or non-differential misclassification, on epidemiological inference typically receive less attention. Most researchers realise that non-differential misclassification of exposure and/or outcome measures will lead to an attenuation of the resulting measure of association.

However, the fact that random measurement error in potential confounding variables may wreak havoc on the inferences which are made from study results is seldom acknowledged. The non-differential misclassification of a dichotomous confounding variable may lead to inadequate statistical adjustment (often referred to as residual confounding) and the false appearance of statistical interaction when none is present.[2] When confounders are measured as polytomous or continuous variables (for example, condom use never/sometimes/always or number of sexual partners), random measurement error can bias the adjusted measure of association unpredictably—in some instances making the adjusted measure of association less accurate than the crude.[3 4] These forms of misclassification are generally of greatest concern when the true exposure-disease association is relatively weak compared with the exposure-confounder and outcome-confounder relation,[5] as is the case in most research around STIs. Even small random errors can have major effects on adjusted measures of association, and the unpredictability of the effects of misclassification may be compounded in multivariate analyses.[6]

With this in mind, Fenton *et al*'s review of the difficulties involved in the accurate measurement of sexual behaviour has powerful implications for studies attempting to control for covariates associated with risk for STIs. Studies which attempt to adjust during statistical analysis for numbers and types of sexual partners, frequency of sexual contacts, or condom use practices, are likely to encounter some degree of random measurement error. Although perhaps non-differential with respect to exposure or outcome, this mismeasurement may lead to unpredictable biases and/or mis-specified analyses, and in turn, spurious inferences.

In summary, the inadequate measurement of sexual behaviour requires special consideration in any study attempting to adjust for the confounding role of sexual behaviours in associations involving STIs. We hope that Fenton *et al*'s review of the challenges posed by the collection of sexual behaviour data helps to draw attention to this frequently overlooked methodological aspect of the epidemiology of STIs.

LANDON MYER
HIV Prevention and Vaccine Research, South African Medical Research Council, PO Box 658, Hlabisa 3937, South Africa

CHELSEA MORRONI
Women's Health Research Unit, Department of Public Health, University of Cape Town, South Africa

Correspondence to: Landon Myer, Fogarty-AITRP, Division of Epidemiology, School of Public Health, Columbia University, 600 West 168th Street, PH 18, New York, New York, 10032, USA

Landon.Myer@mrc.ac.za or landon_myer@hotmail.com

1 Fenton KA, Johnson AM, McManus S, *et al.* Measuring sexual behaviour: methodological challenges in survey research. *Sex Transm Inf* 2001;**77**:84–92.
2 Cox B, Elwood JM. The effect on stratum-specific odds ratios of non-differential misclassification of a dichotomous covariate. *Am J Epidemiol* 1991;**133**:202–7.
3 Wacholder S. When measurement errors correlate with truth: surprising effects of non-differential misclassification. *Epidemiology* 1995;**6**:157–61.
4 Brenner H. Bias due to non-differential misclassification of polytomous confounders. *J Clin Epidemiol* 1993;**46**:57–63.
5 Marshall JR, Hastrup JL. Mismeasurement and the resonance of strong confounders: uncorrelated errors. *Am J Epidemiol* 1996;**143**:1069–78.
6 Phillips AN, Davey G. Bias in relative odds estimation owing to imprecise measurement of correlated exposures. *Statistics in Medicine* 1992;**11**:953–61.

Is *Mycoplasma hominis* a vaginal pathogen?

EDITOR,—We would like to comment on the study by Arya and colleagues[1] in which they failed to find evidence for *Mycoplasma hominis* being pathogenic in the vagina, or otherwise contributing to bacterial vaginosis (BV). They mentioned the 21 year old review of Taylor-Robinson and McCormack[2] who surmised that *M hominis* might act in symbiosis with other organisms or as a sole pathogen in BV. The latter was referred to as non-specific vaginitis or *Gardnerella* associated vaginitis at that time, the term BV being used from about 1984. Since then, much has been learned about the vaginal microflora in health and disease, but the question of which bacteria, if indeed any, cause BV remains unanswered. The few *M hominis* organisms in the healthy vagina appear to behave as commensals. We challenged[3] the suggestion of Mårdh and colleagues that *M hominis* was associated with a number of genital signs and symptoms after BV had been excluded, our assertion being that *M hominis* organisms outside the context of BV would be present in small numbers and, therefore, unlikely to cause a problem. In contrast, the few *M hominis* organisms in the healthy vagina increase in number, perhaps by 10 000-fold or more, in the vagina of women with BV. This increase, however, occurs only late in the development of BV.[4] Indeed, it is rare to find large numbers in the "intermediate" (grade 2) stage between the normal vaginal flora and "full blown" BV (grade 3). Thus, in the study by Arya and colleagues we have difficulty in understanding why only 35 (48%) of the 73 women with *M hominis* positive BV had large numbers of organisms ($>5 \times 10^5$). A Gram stain evaluation should have distinguished women with grade 2 flora from those with grade 3. Be this as it may, the authors contend that because the additional presence of *M hominis* with *G vaginalis* and strict anaerobes did not seem to increase the likelihood of the women developing BV, *M hominis* is not involved. It is clear that *M hominis* organisms are not essential for the development of BV and unlikely that their initial presence in the vagina increases the likelihood of BV developing. However, if they are present in the vagina initially, then they will multiply as indicated and large numbers will ensue. The data of Arya and colleagues do not resolve the issue of whether large numbers contribute to the disease process or are involved in its persistence. Against this, as they point out, is a study[5] in which metronidazole, inactive in vitro against *M hominis,* cleared vaginitis, and doxycycline, active against *M hominis*, did not. However, it should also be remembered that *M hominis* organisms caused pharyngitis and cervical lymphadenopathy when given orally in large numbers to volunteers,[6] indicating the pathogenic potential of the organisms. Furthermore, the *M hominis* species is heterogeneous, some strains having greater epithelial cell adherence properties than others. We do not see any data that point to *M hominis* being a sole pathogen or co-pathogen in the vagina but, equally, we are not convinced by data that purport to show that it is not.

DAVID TAYLOR-ROBINSON
Department of Genitourinary Medicine, Imperial College School of Medicine, St Mary's Hospital, Paddington, London W2 1NY, UK

ISOBEL J ROSENSTEIN
Scientific Development Division, Public Health Laboratory Service, Headquaters Office, 61 Colindale Avenue, London NW9 5DF, UK

1 Arya OP, Tong CYW, Hart CA, *et al.* Is Mycoplasma hominis a vaginal pathogen? *Sex Transm Inf* 2001;**77**:58–62.
2 Taylor-Robinson D, McCormack WM. The genital mycoplasmas. *N Engl J Med* 1980;**302**:1003–10.
3 Taylor-Robinson D, Rosenstein I. Vaginal flora changes associated with Mycoplasma hominis. *Am J Obstet Gynecol* 1998;**178**:415–6.
4 Rosenstein IJ, Morgan DJ, Sheehan M, *et al.* Bacterial vaginosis in pregnancy—distribution of bacterial species in different Gram stain categories of the vaginal flora. *J Med Microbiol* 1996;**45**:120–6.
5 Pheifer TA, Forsyth PS, Durfee MA, *et al.* Nonspecific vaginitis: role of Haemophilus vaginalis and treatment with metronidazole. *N Engl J Med* 1978;**298**:1429–34.
6 Mufson MA, Ludwig WM, Purcell RH, *et al.* Exudative pharyngitis following experimental Mycoplasma hominis type 1 infection. *J Am Med Assoc* 1965;**192**:1146–52.

Response of hepatitis B induced membranoproliferative glomerulonephritis to HAART

EDITOR,—Renal disease occurring in HIV infected individuals is well described.[1 2] HIV associated nephropathy (HIVAN) is the